THE BRADSHAW LECTURE, 1899.

THE CAUSATION OF FUNCTIONAL CARDIAC MURMURS.

FOXWELL.

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ON THE

CAUSATION OF FUNCTIONAL HEART MURMURS.

Dr. Foxwell's arguments and experiments in support of his theory are sound and, to our mind, more convincing than any of those adduced in favour of hypotheses brought forward by other observers. He leaves no part of the subject untouched. He reasons both on physiological and pathological grounds, and his conclusions are difficult to refute.—Extract from a leading Article on the Bradshaw Lecture in the *Lancet* of Nov. 11, 1899.

THE BRADSHAW LECTURE

ON THE

CAUSATION OF FUNCTIONAL HEART MURMURS.

DELIVERED BEFORE THE ROYAL COLLEGE OF PHYSICIANS, OF LONDON,

ON NOVEMBER 2, 1899.

BY

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THE BRADSHAW LECTURE

ON

THE CAUSATION OF FUNCTIONAL HEART MURMURS.

Mr. President and Gentlemen,

Whēn Sir Samuel Wilks graciously surprised me with the request to deliver this lecture I did not hesitate to accept the honour, but for some time lay in doubt as to my subject. In the end I came to think that I should be most likely to have matter worthy your acceptance if I spoke concerning things in which I was most interested. Hence the title of this lecture.

The etiology of the so-called inorganic cardiac murmurs has always seemed to me to be one of the *opprobria medicinæ*. These murmurs are heard by each one of us almost daily, and yet to none can the vague and varying statements of their causation have ever proved satisfactory. During the last 16 years their etiology has been with me a subject of much thought and investigation. At last I feel quite clear in my own mind as to its nature. But to convince you is a different, more arduous, and far more important matter—one which, I perceive, may well be beyond me and one to which I now address myself with a full sense of its difficulty.

In the short space of one lecture I find it impossible to consider duly all these murmurs; I shall therefore confine myself to the commonest and yet most obscure—I mean the murmur heard over the second and third left spaces which, for the sake of brevity, I shall speak of as the pulmonary murmur. This murmur is systolic in time and is never accompanied by a

diastolic element. It is best heard (Fig. 1) over the second and third left spaces close to the sternum, and is best conducted thence up and out towards the left shoulder, travelling along the

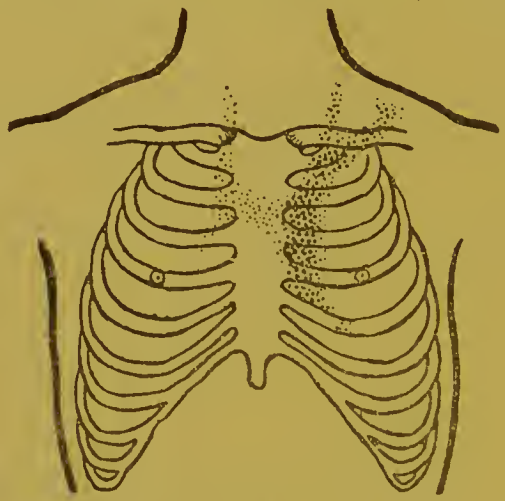


FIG. 1.

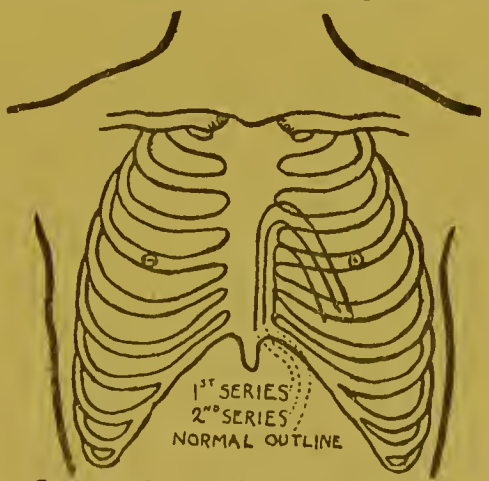
Area of audition of the murmur.

arc of a circle which has the axilla for its centre. It is also sometimes well heard in the fourth left space and may reach the apex, but it does not increase in loudness as it approaches this point, as do the murmurs of mitral incompetence and aortic stenosis. It may cross the sternum, appearing on the right side from the clavicle to the third rib, but it loses greatly in loudness in doing so and never travels far in this direction. When heard to the right of the sternum it is most evident in the second space but may even reach up into the vessels of the neck. In character it is loud, harsh, and blowing, and gives the impression of having a superficial origin. It is never rumbling or purring, thus markedly differing from the murmur of organic pulmonary stenosis. A notable characteristic, considering its harsh loudness, is its want of travelling power; this is far less than in the case of organic murmurs generally, or, indeed, than in that of the functional murmur arising at the mitral orifice. It is not as fugitive as other functional murmurs, but remains with fair constancy till it vanishes for good, though, as the conditions producing it ameliorate, it sometimes requires a few sharp steps, or the supine position, to develop it. As a rule it is more evident

in the supine than the erect posture, especially if it be listened for immediately upon the patient's lying down before the circulation has been able to accommodate itself to its new relation to gravity. So much is this the case that it is doubtful if it be not the dynamic rather than the static change in position which is the more important element in its intensification. This is further borne out by the fact that any quick exertion, even a few sharp steps, will increase it as easily and as much as lying down. At the same time anatomical considerations induce the belief that the supine attitude conduces to its production, as will be seen when the question of dulness is discussed.

Accompanying this murmur we have the following *signs*:—

A. The apex is pushed out and up, in a moderate instance lying beneath the lower part of the fourth space a little to the left of the nipple line. I find from an analysis of 23 cases in which this murmur, *and this murmur alone*, existed, the exact spot of the average apex was the middle of the fifth rib one-fifteenth of an inch within the nipple line. In another series of cases which I published in 1895* I similarly found that the apex was situated at the lower edge of the fifth rib and in the nipple line, so that the position was very nearly the same in the two series. (Fig. 2). In the supine position this removal



OUTLINES OF DEBILITATED HEARTS: THE "AVERAGE" HEART OF THE TWO SERIES OF CASES

FIG. 2.

Outlines of debilitated hearts, the "average" heart of the two series of cases.

* "Essays in Heart and Lung Disease," p. 339.

of the apex up and out is still further increased. Such apical displacement roughly corresponds to a movement along the arc of a circle the centre of which is the aortic valves, probably the most fixed portion of the heart. This only refers to uncomplicated cases of pulmonary murmur; if a mitral or tricuspid be added, then the movement changes in its direction, being more out than up, more in the direction of a tangent touching the circle in the situation of the normal apex than along the corresponding arc. Indeed, the varying dilatation of the two ventricles may be fairly gauged by watching this movement of the apex. (Fig. 3.)

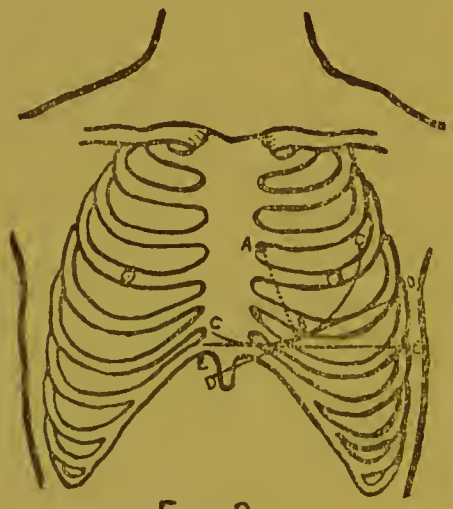


FIG. 3.

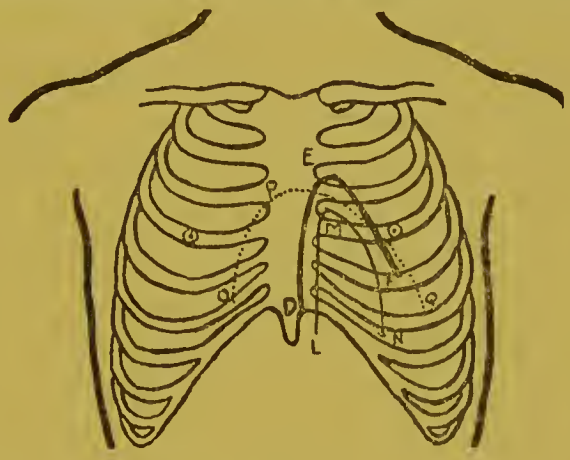
If A = centre of aortic valves = heart's axis of rotation : B = normal apex : C B C = arc of a circle whose radius is A B : D D = tangent at B ; E E = tangent at point between B and C—then if the right ventricle alone enlarge the apex will travel along C B C ; if the left ventricle share in the enlargement slightly, along D D ; if the left ventricle be much enlarged, along such a tangent as E E.

B. The dulness* is little,. if at all, increased to the right (Fig. 2); in the same series I find that it is only three-tenths of an inch to the right of the left edge of the sternum, and in my former series it did not quite reach the mid-sternal line. But the vertical dulness is much raised; instead of being at the upper border of the left fourth cartilage at its junction with the sternum it reaches to the middle of the third cartilage. In

* This always refers to the superficial or absolute dulness. I have elsewhere stated my reasons for preferring this. Op. cit., p. 328.

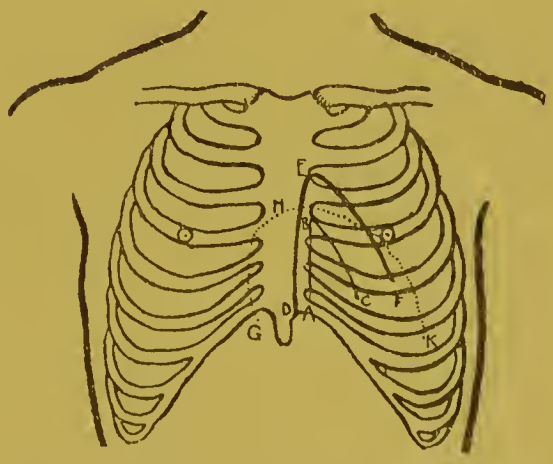
the earlier series it reached even higher, extending into the lower part of the second space.

This peculiar outline of cardiac dulness is characteristic of the debilitated heart and contrasts strongly with that of any heart enlarged by inflammatory disease of the pericardium or valves. Such difference is well exemplified in the accompanying diagrams (Figs 4 and 5) where A B C = outline of



DEF = OUTLINE OF HEART IN DEBILITY.
LMN = " " " " AORTIC DISEASE.
OPQ = " " " " ADHERENT PERICARDIUM

Fig. 4.



DEF = OUTLINE OF HEART IN DEBILITY
ABC = " " " " HEALTH
GHK = " " " " MITRAL DISEASE

Fig. 5.

normal heart, D E F = that of the heart in debility, G H K of a heart with advanced mitral disease, L M N in a case of aortic disease, and O P Q that of pericardial adhesion. This vertical dulness changes considerably with posture, always rising still further when the patient is supine and at the same time extending more to the left of the sternum.

C. Another characteristic sign is the pulsation in the second, third, and fourth left spaces; this pulsation is evident and palpable, has a vermicular movement, and extends from above downwards and outwards towards the apex; occasionally it is accompanied by a feeble thrill of slow and large vibrations, but this never approaches the strength and rolling power of the thrill accompanying organic pulmonary stenosis.

D. Usually there is a slapping accentuation of the second sound; this often heralds the appearance of a murmur and lasts for some time after it has gone. Evidently it is produced by a less degree of the same conditions which originate the murmur.

E. There is often when lying supine an indefinite pulsation in the veins on the right side of the neck; this is not usually present in the erect posture, and disappears when the peripheral venous stream is cut off.

F. The pulse varies considerably; it is nearly always short and ends abruptly, as if the ventricle had suddenly struck work; the onset, also, is too abrupt; the amplitude of the wave during its short existence is usually small, but by no means necessarily so; its diameter may be of quite average size, though the pulse would never be described as large or full. At the same time, it is the abruptness of its onset and ending which is most characteristic.

The pulse wave has a trembling unevenness which can be best described by the adjective "thrilly," and suggests to the mind a ventricle which is labouring and has to perform its systole with a succession of abortive contractions instead of a long and steady one. An attempt is made to make up for this feebleness of individual effort by an increase of rate. In one

case which has come under my notice, with the onset of debility, the pulse-rate has gradually risen from 60 to 80; it is rare for patients with pulmonary murmurs to have a rate below 80. The tension of the impulse is sometimes high and the radial may feel hard during the stroke; but it is rarely correct to speak of the tension generally as high, for the vessel nearly always collapses and feels empty between the beats. Even this modified high tension only occurs in comparatively slight cases and in all the pulse gives the impression that a smaller cylinder of blood than usual is being sent into the aorta at each systole.

Of the *symptoms* which occur with this condition the chief and most evident is dyspnœa on exertion, especially if this exertion be in any way sudden, such as that of running upstairs or vivid emotion. Exertion guardedly approached and slowly undertaken may be performed without any dyspnœa. But no exertion can be long maintained; weariness, physical and mental, comes very quickly. Palpitation, peevish irritability, and dyspepsia are common. The skin is dull, toneless, and pallid. This pulmonary murmur very often occurs during adolescence and is especially common in chlorotic girls. On the other hand it is very rarely found after 60 years of age, but it not seldom appears between the ages of 40 and 50. When it arises towards middle life it is most difficult to remove; when it occurs in adolescence without anæmia it resists treatment more than when anæmia is present, it vanishing most speedily when chlorosis exists.

The patients often give you a history of cardiac strain—several of mine have been sprinters on the cycle track or running path, or else they have returned to full work too soon after an exhausting illness, or they have been subjected to prolonged anxiety or mental strain. Most commonly their condition is the result of a gradual lowering of the constitutional state—they have slowly fallen below par.

In examining the heart *in situ after death* we find the situation of the apex changed as was indicated during life; there is no enlargement of the right or left auricle, nor does the right ventricle extend unduly to the right; but sometimes it forms the

apex and may quite hide the left ventricle. But what is most interesting is the enlargement upwards of the right ventricle; this dilatation is almost entirely confined to the conus arteriosus. On removing the carapace the conus not only bulges unduly forward but is wider than normal; moreover, the pulmonary artery is similarly widened and bulging forward. Here is evidently to be found the explanation of the increased vertical dulness on the left of the sternum just as we similarly find it on the *right* of the sternum in cases of aortic dilatation from chronic aortitis. The stretching of the conus carries the pulmonary valves upwards, and in 20 cases of exhausting disease, reported by various observers, they were found on the average under the second costal cartilage. These valves after death are usually found higher up than was the upper limit of absolute dulness during life.

The following additional cases further exemplify this condition. The *post-mortem* statements were made by Dr. Douglas Stanley, our pathologist, and the cardiac outlines during life were taken from the clinical notes of each case, such notes being made by different physicians and house physicians.

Case 1.—The patient was a man, aged 39 years. Death resulted from cholangitis and duodenal cancer. After death the pulmonary artery was found to reach up to the lower edge of the second cartilage and it extended from the mid-sternum for one and a half inches to the left of this bone (=width of two and a quarter inches). The pulmonary valves occupied the middle of the second space. The apex was situated at the upper border of the fifth rib just inside the left vertical nipple line.

Case 2.—The patient was a man, aged 36 years. Enteric fever was the cause of death. After death the pulmonary valves were seen to lie beneath the lower edge of second left cartilage. The apex was situated beneath the fifth rib, one eighth of an inch internal to the left vertical nipple line. During life, according to the only note made in this case, when the patient was admitted to hospital, the apex beat reached to the fourth space, half an inch internal to the left vertical nipple line. The vertical

dulness was at the upper border of the fourth cartilage and the right dulness at the left edge of the sternum. No murmurs were heard.

Case 3.—The patient, a male, aged 19 years, died from purpuric anæmia. During life the apex beat reached to the lower border of the fourth space. There was right dulness at the left edge of the sternum and vertical dulness at the third upper border of the cartilage. Soft systolic murmurs were heard at the apex beat and in the pulmonary area. After death the apex was in the fourth space and left vertical nipple line, the right margin was two and three-quarter inches from the mid-sternum, and the pulmonary valves lay under the second cartilage.

Case 4.—The patient, a man, aged 47 years, had suffered from cirrhosis of the liver with hæmatemesis. During life the apex beat was in the fourth space, one and a quarter inches without the left vertical nipple line. There was right dulness at the left edge of the sternum and vertical dulness at the lower border of the second space. A systolic murmur heard in the first four left spaces was most marked in the second and third spaces. After death the apex was in the fourth space, three and a half inches from the mid-line. There were right dulness one and a quarter inches from the mid-line and vertical dulness at the lower border of the second space. The left pleura was very widely adherent.

Case 5.—The patient, a male, aged 16 years, had died from anæmia. After death the apex was in the fourth space and left vertical nipple line. The heart reached one and a quarter inches to the right of the mid-sternum at the fourth cartilage. The pulmonary valves lay beneath the middle of the left first space. Before death the apex beat was slightly internal to the left vertical nipple line in the fourth space. Systolic murmurs were heard at the apex beat and in the pulmonary area, and a venous hum was audible in the neck.

Case 6.—The patient had died from phthisis. The pericardium was not at all adherent on the right but it was universally so on the left. The sac reached from the first left

cartilage to the position of the apex beat in the fourth space. The left ventricle was not enlarged. The apex beat was formed by the right, which reached some eighth of an inch below the left. The posterior pulmonary valve was one-third of an inch above the anterior aortic; the anterior pulmonary valve was half an inch above its posterior fellow, and therefore five-sixths of an inch above the aortic valves. This case shows in a marked manner that the anterior wall of the pulmonary artery rises more than does the posterior wall.

The foregoing is a brief statement of the chief conditions found during life and after death in cases of debilitated heart. They are so frequently associated that there must be a causal relationship between them, and any explanation of one should also help to explain the rest. An explanation of the cause of the pulmonary murmur is the more likely therefore to be the true one if it also explains the origin of the other conditions described. What, then, is its explanation?

I.—It cannot be due to the quality of the blood for the following reasons:—

1. In many cases where this is greatly changed there is no murmur. The following may be cited as a few examples:—

Case 7.—The patient suffered from chlorosis. Red corpuscles, 3,700,000; hæmoglobin, 65 per cent., with slight poikilocytosis. No murmurs were heard.

Case 8.—A man, aged 61 years, was the subject of carcinoma of the stomach. Red corpuscles, 3,625,000; hæmoglobin, 62 per cent. No murmurs were heard.

Case 9.—The patient was a male who suffered from phthisis of both lungs. Red corpuscles, 2,910,000; hæmoglobin, 45 per cent. The corpuscles varied much in size. No murmurs were heard.

Case 10.—A man, aged 21 years, had hectic fever after operation. Red corpuscles, 3,600,000; hæmoglobin, 72 per cent. No murmurs were heard.

Case 11.—The patient was a male who was the subject of subpectoral abscess. Red corpuscles, 4,000,000; hæmoglobin,

68 per cent. The cells were very pale: there was some poikilocytosis. No murmurs were heard.

Case 12.—The patient, a female, aged 18 years, suffered from gastric ulcer and hæmoptysis. Red corpuscles, 2,470,000; hæmoglobin, 20 per cent. There were many microcytes and some poikilocytosis. No murmurs were heard.

Case 13.—The patient, a man, aged 40 years, was the subject of plumbism. Red corpuscles, 3,600,000; hæmoglobin, 40 per cent. No murmurs were heard.

Case 14.—The patient, aged 24 years, suffered from Hodgkin's disease. Red corpuscles, 4,770,000; white corpuscles, 10,000; hæmoglobin, 33 per cent. No murmurs were heard.

Case 15.—The patient was a man, aged 35 years, who suffered from malaria. Red corpuscles. 3,820,000; hæmoglobin, 48 per cent.; there was some poikilocytosis. No murmurs were heard.

Case 16.—The patient, a man, aged 41 years, suffered from pernicious anæmia. Red corpuscles, 1,320,000; hæmoglobin, 30 per cent. There were many microcytes, some megalocytes, and much poikilocytosis. No murmurs were heard. This man was an in-patient, and had been examined many times.

Case 17.—The patient, a female, aged six years, was the subject of abdominal abscess. Red corpuscles, 3,814,200; hæmoglobin, 55 per cent. No murmurs were heard.

2. Again, in addition to being absent when the blood is much deteriorated, the murmur frequently exists when there is no change in the blood. And in this regard I would ask you to note that whilst many of Class 1 were in-patients and lying in bed, yet no example of the present class could be obtained from such cases. This point I shall discuss later, but I would ask you to bear it in mind, for in my judgment it is strong evidence that the murmur is due to a special form of cardiac strain. Amongst out-patients the following are examples.

Case 18.—The patient, a youth, aged 18 years, convalescent from a long attack of typhoid fever, was up and about. Red corpuscles, 4,610,000; hæmoglobin, 82 per cent. There was much poikilocytosis. A pulmonary murmur was well heard

both when the patient stood erect and (much louder) when he assumed the supine position.

Case 19.—The patient was a woman, aged 40 years, who suffered from debility. Red corpuscles, 4,500,000; hæmoglobin, 81 per cent.; slight poikilocytosis. A pulmonary murmur was well marked.

Case 20.—The patient, a female, aged 20 years, the subject of debility, was under treatment for four months. Red corpuscles, 5,400,000; hæmoglobin, 81 per cent. There were a few microcytes (two examinations were made). A pulmonary murmur was heard on each of three examinations.

Case 21.—The patient, a girl, aged 17 years, suffered from debility. Red corpuscles, 4,855,000; hæmoglobin, 86 per cent. A pulmonary murmur was well marked.

Case 22.—The patient, a male, aged 21 years, suffered from debility. Red corpuscles, 5,100,000; hæmoglobin, 100 per cent.; the cells were quite normal. A pulmonary murmur was only heard when the patient was in the supine position.

Case 23.—The patient, a youth, aged 18 years, was the subject of debility. Red corpuscles, 5,400,000; hæmoglobin, 94·5 per cent. (the mean of four examinations). A pulmonary murmur was heard when either the erect or supine position was assumed.

Case 24.—The patient, a female, aged 18 years, suffered from debility. Red corpuscles, 4,690,000; hæmoglobin, 81 per cent.; slight poikilocytosis, fairly uniform in size. A pulmonary murmur could be heard when the patient was erect or when she was supine.

Case 25.—The patient, a man, aged 20 years, was debilitated. Red corpuscles, 5,150,000; hæmoglobin, 90 per cent. The cells were normal. There was a well-marked pulmonary murmur to be heard when the patient was lying down.

Amongst my private patients I find a considerable number of such cases, but I cannot vouch for the condition of their blood as I did not examine this. All I can say is that their appearance did not give the slightest indication of poor blood. It may perhaps be worth while to give one or two instances.

Case 26.—The patient, a man, aged 22 years, had "always gone in much for athletics." For the last two years he had felt an "indigestion lump" behind the sternum when racing. He was fairly fit on rising in the morning, but he would soon get "all gone," palpitation easily coming, especially after dinner. He was well set up and strongly built, and showed no sign of anæmia. The apex beat reached the fifth space and nipple line; the impulse was diffused. There was vertical dulness at the third space and right dulness at the left edge of the sternum In the second and third left spaces was visible pulsation with a loud systolic murmur. This was well conducted out and up into the first space and towards the shoulder. It was also heard in the fourth and fifth spaces, nearly reaching the apex.

Case 27.—The patient, a man, aged 23 years, had strained his heart at Cambridge, three years previously, at football. He gave up active exercise, but strained his heart again at Folkestone a year later whilst cycling. Eighteen months afterwards his heart again went wrong when tea-planting in Ceylon, his symptoms being dyspnœa and cold extremities. He sought my advice for violent earache, from which he had suffered for three days. He had no sign of anæmia and was well-built. The apex-beat was in the fifth space, half an inch outside the left vertical nipple line. There was vertical dulness in the third space and right dulness at the left edge of the sternum. A short systolic murmur was heard in the second space on lying down.

Case 28.—The patient was a man, aged 27 years. Formerly he had been very athletic, but when seen he was depressed, neurotic, and easily subject to palpitation. He did not look anæmic, but his blood was not examined. He came to be tested for insurance. The apex beat was in the normal situation, the impulse slapping and thumping. The second pulmonary sound was accentuated. There was a systolic murmur, which was best heard in the second left space, half an inch from the sternum, thence into the first space and out to the shoulder, also feebly in the third space, not over or to the right of the sternum. As a rule no murmur was heard in the third and

fourth left spaces, but an occasional systolic murmur was heard at, and a little outside of, the apex beat. There was vertical dulness at the third cartilage and right dulness at the mid-sternum.

Case 29.—The patient, a youth, aged 17 years, had never been very robust, but he entered into games well. There was no evidence of anæmia. He sought advice because he did not feel up to his work. The apex beat reached the fifth rib and the left vertical nipple lfne. There was vertical dulness in the third space and right dulness at the left edge of the sternum. A systolic murmur sprang up in the second, third, and fourth left spaces on sharply walking or on lying down.

3. If it were due to changes in the blood then similar murmurs should arise in all parts of the body to a greater or less extent, especially where there are angles or sharp curves; but there are no such murmurs.

4. Immediately after severe hæmorrhage such generalised murmurs do arise, these cease in a day or so when the vessels are again sufficiently distended with (what must be) hydræmic blood, but later, if debility sets in as a result of the hæmorrhage, the pulmonary murmur appears and persists till the patient is restored to his customary vigour.

5. Persons convalescing from exhausting diseases—*e.g.*, typhoid fever—whether suffering from anæmia or not, may have no murmur so long as they are confined to bed, but when sufficiently recovered to be up and about a murmur not seldom arises. An instance of this is Case 18, but a very interesting and to my mind more convincing example is the following:—

Case 30.—A married woman, aged 29 years of age, was admitted to hospital with enteric fever. Seven days prior to her discharge, before she had been allowed up, the following was the note made of her heart:—The apex beat reached the fourth space, half an inch outside the left vertical nipple line. There was vertical dulness over the upper border of the third left cartilage and right dulness to the right of the left edge of the sternum. A harsh systolic murmur was heard at the apex beat

and a short soft whiff at the beginning of systole in the second left space. Three weeks later, after the patient had done her housework for a fortnight, another observer, quite ignorant of the previous note, made the following independent observations :—The apex beat reached the lower border of the fourth space in the left vertical nipple line. There was vertical dulness at the upper edge of the third left cartilage and right dulness a quarter of an inch to the right of the left edge of the sternum. There was no apical systolic murmur, but a well-marked systolic murmur in the second left space which was well conducted up and out into the first; it was also slightly heard in the third left space. It became louder on lying down. A reference to Fig. 6 explains what happened.

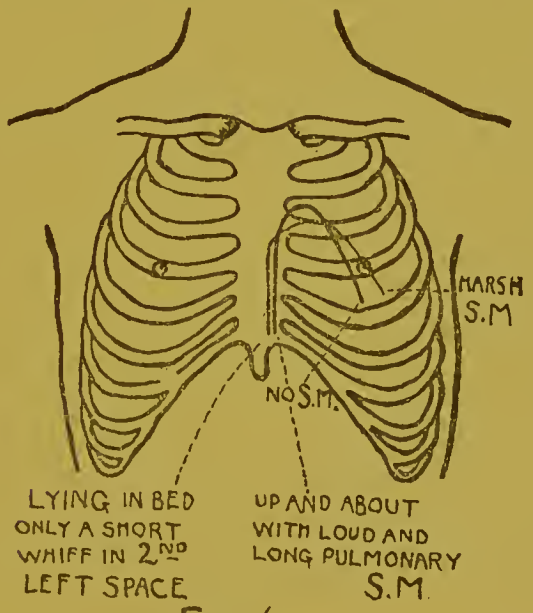


FIG. 6.

Lying in bed only a short whiff in the second left space, but harsh systolic murmur at apex. Up and about loud and long pulmonary systolic murmur, but no apical murmur.

The improvement in the general health and strength pulled the heart together; the dilated left ventricle came back to its normal size, as shown by the recession of the apex beat, and with this the mitral valve ceased to leak; but the conus was not able to withstand the strain put upon it by all the short little exertions

of housewifery, hence the pulmonary murmur became much louder and the vertical dulness still further raised, although the lungs were no doubt more fully inflated than when the patient was confined to bed. The blood of this patient was not examined, as she gave no evidence at any time of anæmia, though no doubt its quality was somewhat below par on both occasions.

6. By a method presently to be described various fluids were forced through the human heart with a view to determine whether changes in the circulating medium made any differences in the sounds produced.

Experiment 1.—The first heart experimented upon was that of a woman, 36 years of age, who died from gangrene of the leg with granular kidneys. The pericardium was healthy but the whole heart was enlarged. The right ventricle was dilated and its wall was chiefly composed of fat, its muscular layer being extremely thin ($\frac{1}{32}$ inch.) The left ventricle was dilated and hypertrophied, the wall being three-quarters of an inch thick but showing extensive fatty degeneration. The mitral and aortic valves were thickened, the aorta was atheromatous, and the endocardium was thickened and opaque. This heart, then, was peculiarly suited to increase the murmur-producing capacity of any fluid. The pericardium (parietal layer) was kept on, ordinary tap-water was first used, and the sounds at the base of the heart were carefully noted. These were unchanged when a saline solution (specific gravity 1045) was passed through. They still remained unchanged when a thick solution of boiled starch, containing lumps half an inch square and one-sixteenth of an inch thick, was employed. It was particularly noted that even if several of these lumps passed together yet no change in the sounds occurred. Yet if pressure were made by the stethoscope a murmur appeared and remained unchanged in quality on again passing the three fluids through. A window was then cut in the parietal pericardium over the pulmonary artery and conus, through which these bulged, and the same three fluids were used. With each systole a short murmur was

heard and this remained the same whichever fluid was used. This experiment shows that certain changes in the circulating medium produce no change in the sounds, but certain changes in the shape of the containing apparatus induce a murmur.

Experiment 2.—This experiment was made upon a heart from a healthy man, aged 32 years, who died from fractured base. The heart was healthy. (*a*) With tap-water. A soft murmur was heard over the aorta as well as over the pulmonary artery. This was unexpected and in seeking for the cause a portion of the pulmonary artery next the aorta was found to be bulging and uncovered by pericardium. The torn pericardium was brought together and sewn up over the bulge. After this was done no murmur was heard. (*b*) Salt solution (specific gravity 1035) was next passed through. The sounds remained unchanged. (*c*) A solution of milk and borax (specific gravity 1056) was used. Again there was no change in the sounds. (*d*) A solution of starch (specific gravity 1005). No murmurs were heard, but the sounds generally were deadened, and it was more difficult to produce a murmur by locally compressing the conus or pulmonary artery. (*e*) A window was now made in the pericardium over the pulmonary artery and conus, allowing these to bulge through it. With the same starch solution a short soft murmur was heard, but this could not be detected with the stethoscope on the aorta. (*f*) Tap-water was now used and the murmur become louder and could be detected over the aorta. (*g*) The pericardium was entirely removed; tap-water was again used; no murmur could be detected. In this experiment it must be noted that starch solution tended to prevent the production of a murmur. But the difference between the viscidity of tap-water and a starch solution—which was made as thick as we could pass through the tubing—is so many times greater than that which could arise between varying states of the blood that much stress cannot be placed upon it. Still, so far as it goes, it is against rather than for the hæmic origin of the murmur, as the great excess of white corpuscles in leukæmia must surely increase the viscidity of the blood in these cases, and therefore militate

against the production of the murmurs which are usually heard in this disease.

7. It has been suggested that as in anæmia the corpuscles are lighter than normal they may occupy the upper semi-circumference of an artery and so tend to produce a murmur by their friction against the sides of the vessels. But in anæmia the specific gravity of the blood serum is also lessened, and there is no evidence to show that the two diminutions do not proceed *pari passu*, and hence there would be no tendency for the corpuscles to deviate from their usual axial position. Moreover, even if true, this could only account for the murmur in cases of anæmia and not for that which arises in other forms of cardiac debility.

For these seven reasons I think it impossible that the murmur can be due to changes in the blood.

II.—If the murmur be not due to a change in the contained fluid it must arise from some defect in the containing apparatus. As its area of audition is localised it would seem that the change must be also localised. That is, it must occur in the heart or some one or more of the great vessels close to the heart.

1. Is it due to regurgitation through the mitral orifice? Against this are these facts: (*a*) Its area of audition is very different from that of an organic mitral lesion; it is feebly, if at all, heard at the apex and is never conducted from the apex to the axilla. When an organic mitral murmur is heard in the second and third left spaces it is evidently a murmur conducted from the apex with gradually lessening intensity; it is true it occasionally becomes a little louder at the base, just as an aortic regurgitant murmur springs up again at the apex, but even then it is not better heard on the left side than on the right, if as well. No one has heard the murmur of organic mitral regurgitation in the second and third left spaces only and failed to discover it at the apex or between this and the axilla. But these two spaces are the very ones in which the murmur whose origin is now being discussed is so frequently alone audible. (*b*) The area of dulness both in functional and organic uncomplicated mitral regurgitation is the same, and very different from that

occurring in cases of uncomplicated pulmonary murmur. (*c*) A functional murmur does arise whose distribution is identical with that of organic mitral regurgitation and is accompanied by similar changes in the area of dulness and situation of the apex, and when it arises in conjunction with the murmur under discussion it tends to modify these in the directions indicated. This functional murmur is recognised as being due to functional mitral leakage; it is very fleeting and is softer than the pulmonary murmur, though much better conducted. It is conducted to the back in the region of the angle of the scapula; the pulmonary murmur is heard with extreme rarity posteriorly; when it is so heard it is at a point in the left inter-scapular region near the spine of the scapula. It is, to say the least, difficult so to arrange one's mind as to believe two sounds so different are due to the same cause.

2. Is it due to tricuspid leakage? Here, again, we can fortunately compare it with a murmur arising from tricuspid leakage due to back pressure in organic mitral disease. This murmur is heard over the lower part of the sternum and about equally in the third, fourth, and fifth spaces on either side of this bone; sometimes it is conducted up into the second right space and even reaches the right clavicle, but this upward conduction does not occur on the left side of the sternum. The right dulness is considerably increased but the vertical dulness scarcely at all. The apex is pushed out to the left and down rather than up. There is a distinct systolic pulsation in the veins of the neck which is best demonstrated by cutting off the peripheral flow. The indefinite undulation which may occur with the pulmonary murmur tends to be presystolic and, at any rate, cannot be definitely connected with the systole, and often vanishes when the peripheral flow is cut off. Finally, in the tricuspid regurgitation due to mitral disease there may be hepatic pulsation, which never arises in the merely debilitated heart.

Beyond all this a purely functional murmur is sometimes heard in cases of debility which has all the characteristics I have just mentioned, and which is, so far as I know, universally allowed to be due to leakage through the tricuspid valve.

Moreover, when in a debilitated subject, in addition to the usual murmur over the pulmonary valves, another springs up having the location I have just described, then the shape of the cardiac outline also changes, widening out on both sides, but not rising any further upwards. The outline takes on, in fact, the shape due to tricuspid leakage as well as that caused by dilatation of the conus. Venous pulsation also arises and we have every reason to believe that we are dealing with a fresh lesion in the shape of a failing tricuspid valve.

3. Is it due to any aortic defect? Virchow's congenital narrowing is only stated as occurring in chlorosis and must be an extremely rare event in this disease. No constriction or dilatation was noted in any case of which I have records, neither of valves nor artery. When degeneration arises in middle life, and chronic aortitis, with its roughening of the inner coat and widening of the aorta, produces a systolic murmur, the clinical signs are very different and the murmur has quite a different habitation.

4. Is it due to a defect in the pulmonary valves? Nowhere can I find in the *post-mortem* notes on individuals whose hearts before death gave evidence of the pulmonary murmur, and this alone, any statement of thickening, constriction, or erosion of these valves. Besides, the murmur differs in many ways from that of organic valvular stenosis; it is far less well-conducted, there is no true purring thrill, and the forcible heave of a hypertrophied right ventricle is entirely lacking.

III.—There is one *post-mortem* abnormality, which is constantly found in patients who have died with this functional pulmonary murmur—that is, dilatation of the conus and pulmonary artery with a carrying up of the root of the artery, so that in the supine condition it tends to lie directly over its bifurcation.* Is this the cause of the murmur? I believe it is and for the following reasons:—

1. The murmur is systolic only; there is never a diastolic portion: hence the orifice of the valve cannot be dilated enough to allow of leakage. *Post-mortem* evidence bears this out, for there

* *Vide* Cases 3 and 4 supra.

was no increase in size in two valves which I measured. We have, therefore, a tube with an annular constriction (Fig. 7),

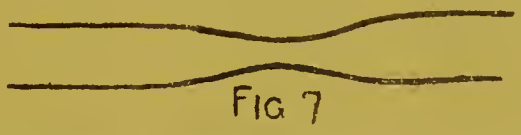


FIG 7

a condition well known as able to induce a murmur.

2. The dilatation of the conus carries the pulmonary artery upwards till the valves are vertically over the bifurcation, which is a fixed point (Fig. 8)—*i.e.*, it lessens the distance between the

FIG 8.

two ends of the artery; the artery therefore tends to fold itself longitudinally, as does the curly brachial in cases of arterial degeneration. This lack of longitudinal tension to a great extent renders the restraining force of the longitudinal fibres of no effect; hence dilatation, being now prevented by the circular fibres only, is much more likely to occur and a pouch-like distension to be produced by the systolic inrush of blood; hence eddies and a murmur.

3. The same want of room and laxness will increase the artery's tendency to flatten itself against the comparatively rigid chest wall and aortic arch, this flattening acting as a partial constriction and so again tending to induce eddies and a murmur.

4. The anterior wall of the pulmonary artery is carried up farther than the posterior wall. One would expect the anterior muscular portion of the conus to stretch and not its posterior ligamentous surface. Moreover, the union of the posterior wall of the pulmonary artery to the aorta would prevent upward movement of the former. Case 6 shows that this actually takes place, the anterior segment of the pulmonary valve being in this

instance some half an inch higher than the posterior one. The plane of the valve ring is, therefore, not perpendicular to the longitudinal axis of the artery and the systolic inrush of blood would tend to direct itself against the side of the vessel and thus induce an eddy and a murmur; further, the anterior wall is also more shortened than the posterior wall and would be laxer and tend more to sag. In fact, the systolic rush of blood would tend to take the direction indicated by the arrows in the accompanying diagram (Fig. 9), a direction evidently murmur-producing.

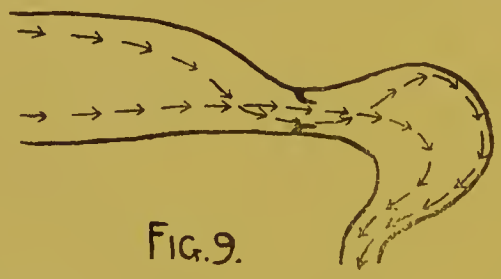


FIG. 9.

5. The movement of the apex upwards and to the left still further alters the angle at which the ventricle lies to the pulmonary artery. This movement of itself would appear to be sometimes capable of inducing a murmur, as the murmur which arises with a left pleural effusion and vanishes *at once* on the withdrawal of the fluid would tend to show.

6. This view would easily explain the increase of the murmur in the supine position. When erect the weight of the heart tends to keep the pulmonary artery and conus on the stretch and so to prevent or lessen their dilatation. In the erect position the ventricle has to force the blood into the artery against gravity and would therefore do so with less power. Lastly, as the systemic tension is lower in the erect than in the supine posture, probably this is also true of the pulmonary circulation; hence the pulmonary artery and conus would be less stretched in the erect posture.

7. It would evidently explain the outline of dulness and also the position of the apex; for if the dilated conus pushes the pulmonary valves upwards it must also equally push the ventricle below it down on the unyielding tendinous portion of the

diaphragm, and thus, after the manner of a see-saw, push the apical part up and out.

So far, then, I have given you direct as well as circumstantial evidence that the murmur is not due to any changes in the blood, and I trust that this evidence will prove satisfactory to you; but I have only been able to advance circumstantial evidence that it is due to a dilatation of the conus and pulmonary artery. To my mind, though circumstantial, this evidence is so strong that I have for long given this explanation of the murmur to students, but feeling that direct evidence is much more convincing I have this year endeavoured to obtain such.

I set myself three questions to answer: First, can this murmur be always heard after death in a heart over which it was heard during life? Secondly, is it never heard after death in the healthy heart? And, thirdly, can the murmur be produced after death by locally dilating the conus and pulmonary artery of a healthy heart?

To these three questions I believe I have obtained affirmative answers which I will now submit to you for your consideration.*

First, let me describe the method of procedure. After removing the carapace from a cadaver on the *post-mortem* table the large vessels were cut as long as possible and then the heart and pericardium were removed together, great care being taken not to puncture the latter organ. The heart thus isolated was placed upon a wooden block on a *post-mortem* table (*vide* diagram Fig. 10, and photograph of the apparatus, Fig. 11). Into the superior vena cava and into a pulmonary vein were fastened two leaden pipes having an internal diameter of one inch, the ligatures including the pericardium. At the other end of each

* I would here gladly acknowledge the great help which my able assistant and pupil, Mr. W. Billington, has given me; to his skill and ingenuity the success of this investigation is largely due. I would also heartily thank my colleagues, both medical and surgical, for the *carte blanche* way in which they gave over to me any hearts at their disposal. I feel greatly indebted to the forbearance of our pathologist, Dr. Douglas Stanley, for allowing me to use the *post-mortem* room as a laboratory for so long a period, and to the enthusiastic help and scientific knowledge of my house physician, Mr. W. H. Coltart. I indeed owe much.

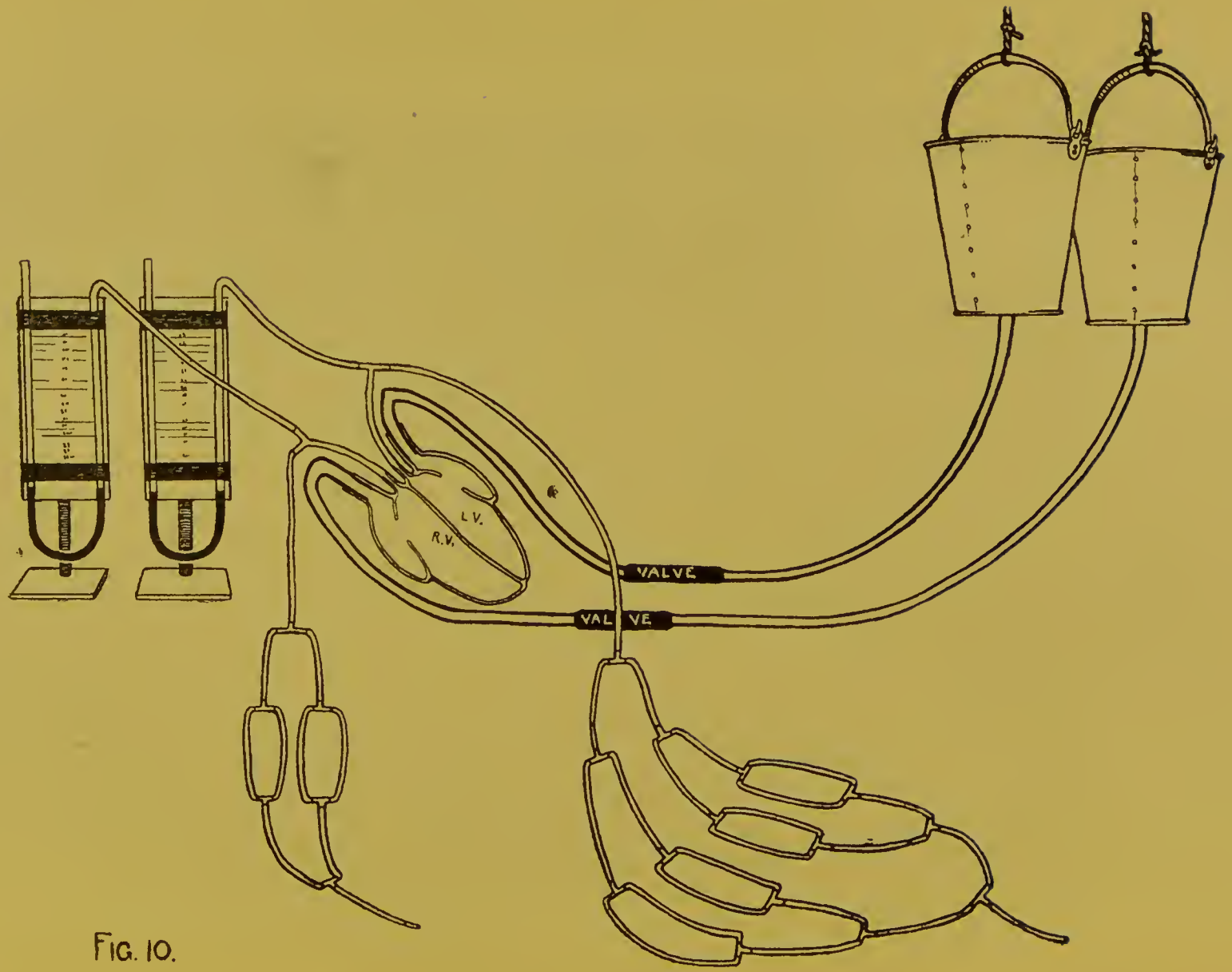


FIG. 10.

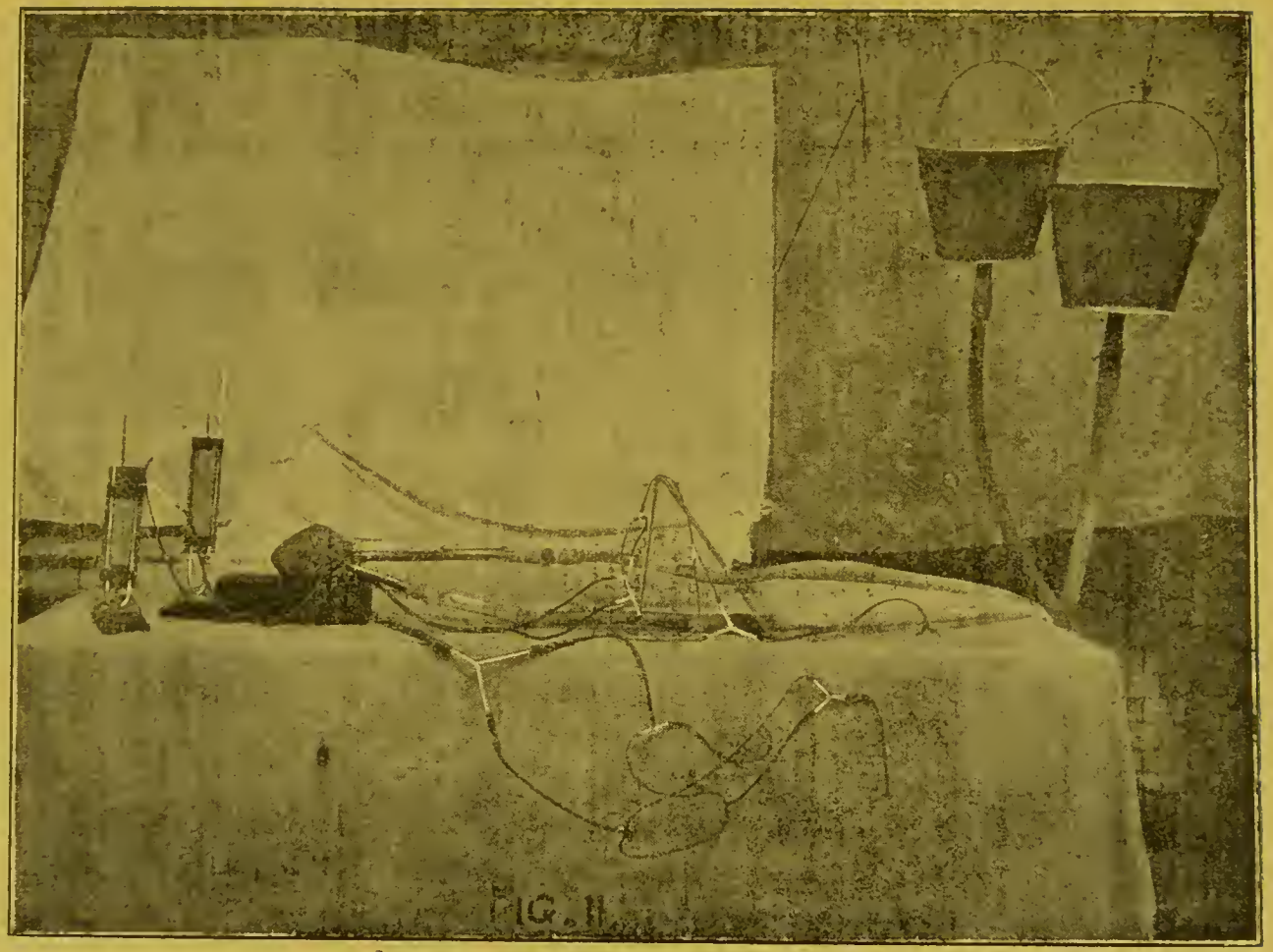


FIG. 11.

of these rigid tubes was a valve which prevented any back flow from the heart, thus acting as mitral and tricuspid valves respectively. Beyond the valves stout rubber tubing of the same diameter led to two buckets which were suspended on pulleys, so that they could be lowered or raised at will and thus vary the venous supply pressure. The buckets were kept full by connection with water-taps. Into the aorta and one limb of the pulmonary artery were similarly fastened two glass T tubes, that in the pulmonary artery having a diameter of two-thirds of an inch and that in the aorta of half an inch. One limb of each T was connected with a mercurial manometer and the other was passed into a series of lessening and bifurcating tubes to represent the resistance of the pulmonary and systemic circulations. It will be noted that the bore of the glass T's is less than that of the arteries; it may be thought that such diminution would tend to induce a murmur; yet as the bore of the aortic T is less than that of the pulmonary, such tendency would be greater in the aorta, yet in no instance was there evidence of such tendency in this vessel, so that this contraction of bore had no effect on the result. When everything was fixed in position the heart was firmly grasped in the two hands of one of us and forcibly compressed, whilst another listened through a binaural stethoscope applied to the heart.

There are three objections to this method. 1. The auricles are subjected to the same systolic pressure as the ventricles and thus occupy too much space during systole; but as each auricle and ventricle practically corresponded to one ventricle the auriculo-ventricular valves being outside the heart, this defect was of small moment. 2. The difficulty of exerting different pressures on the left and right side; we were surprised at the ease with which this was overcome; with a comparatively small experience it was quite possible to make the manometer connected with the aorta average twice the pressure of that connected with the pulmonary artery. 3. The passivity of the heart. This acted in two ways: (*a*) the venous inflow had to distend inert cavities, hence the diastole took far longer than during life unless

we made the venous pressure absurdly large; and (*b*) the septum being lifeless it was pushed into the right ventricle, when the pressure in the left heart greatly exceeded that in the right, and thus tended to occlude the conus. We were therefore compelled to work with pressures of about 80 millimetres and 140 millimetres on the right and left sides respectively when we used both sides together, but we corrected our results by trying the left side alone at 200 millimetres. We found that no tendency to murmur arose in the aorta on doing so.

On the other hand, there were two circumstances which militated against the production of a pulmonary murmur after death by our method as compared with the condition of the heart during life. First, there was nothing to compress the dilated artery and conus, no rigid thoracic wall or aortic arch; and secondly, the weight of the glass T tubes, which hung more or less perpendicularly, tended to stretch the arteries and thus to prevent any attempt at bagging. The first of these conditions we soon learnt the importance of, for any compression of the vessel by the stethoscope easily intensified the murmur.

Such was the method finally adopted, but many varieties of apparatus were first attempted. Most of these were too crude to give trustworthy results and I have not included the experiments made with them in the following list. At the same time I would say that they all bore out the same conclusion and not one of them gave a contradictory result.

One variation in the apparatus I must describe, as with it the first six experiments here recorded were performed. In this two easily compressed pieces of rubber tube took the place of the valves and these were constricted by pressing the edge of a piece of wood upon both with one's hands from 60 to 80 times a minute, the operator being guided by a watch laid on the table. Whilst the constriction was made diastole occurred, and systole took place when it was removed. It is evident that the systolic pressure was now obtained by the weight of water from the buckets, and these were raised to equal 80 millimetres and 200 millimetres of mercury respectively. There were two objections to this method: first, the heart was never emptied and

neither set of semilunar valves could be brought into play; and secondly, and most importantly from the murmur point of view, each ventricle and artery tended to become one dilated tube and a murmur due to local bulging would occur less readily than when the ventricle was steadily decreasing in size as the systole continued.

I regret to say that even including these six I have a very small number of experiments to present to you. Were it not that they all point the same way I should have hesitated to bring them forward at all. But it has not been an easy investigation to take up; the hearts had to be free from valvular disease and had to be made use of as soon as they chanced to appear, which did not always coincide with a period of leisure; moreover, each experiment took three hours in its performance. Not that I in any way grudge the time for, apart from the purpose in hand, it has amply repaid me by the intimate knowledge which it has given me of the potentialities of the heart under varying conditions of strain. With this end alone in view it would be well worth the while of any young physician to repeat them.

Generally, we were assisted in our observations by one or more of the hospital residents or senior students and, to remove as far as possible all tendency to bias, no one stated his opinion till all had examined, and mine was stated last. Further, as we were generally ignorant of the cardiac condition before death, we were able to maintain open minds. The method employed was not capable of investigating tricuspid or mitral murmurs, but incidentally it convinced us that pressure upon the pulmonary artery by the left auricle, with a murmur arising from the constriction of the artery, was a practical impossibility so long as the pericardium remained intact, for even when the auricle was subjected for a considerable time to systolic pressure approaching 200 millimetres it never dilated sufficiently to encroach upon the artery; not even when, as I have stated above, the difference of pressure was enough to so displace the septum ventriculorum as to occlude the conus.

Another collateral result was the negative evidence obtained as to the origin of the murmur in the aorta. Several times we experimented with the left side only and endeavoured to produce a murmur by subjecting it to pressures of over 200 millimetres Hg, using for this purpose hearts several days after their removal from the cadaver, but we never succeeded in gaining any dilatation approaching that of the pulmonary artery, and only once obtained a murmur. Curiously, this murmur was the single exception which proves the rule, for (*vide* Experiment 21) on examining the aorta and its valves we found both the subject of aortitis, so that the murmur was no doubt an organic and not a functional sound.

Perhaps the greatest revelation which these experiments gave us was evidence of the manifold uses of the pericardium, especially the importance of its action as a cardiac support. We divested a sheep's heart of its pericardium and three hours after death it withstood the usual pressure (80 millimetres Hg on the right side) well, but 17 hours later it dilated so greatly and leaked so much through the substance of the right auricle and right ventricle that it was of no use for experimental purposes. The same dilatation and leakage had occurred with the human hearts which we had previously employed. The experiments did not, as a rule, take place within 20 hours of death, yet we thought that the breakdown might be due to disease and not simply to the death of the tissue. But after trying with one or two sheeps' hearts and finding that they gave way equally with those of human beings we kept the heart in its pericardium and were surprised to find its condition maintained for two or three days after death. We were thus at once provided with a ready way of producing dilatation of any portion—viz., by making a window in the pericardium at the spot and allowing the heart substance to bulge through the opening. When such a window was made over the aorta not much bulging and no murmur arose, but if a similar opening was cut over the conus and pulmonary artery considerable bulging always occurred and usually a murmur was developed, whether this had existed previously or no.

The hearts we experimented with may be divided into three classes: (1) those which showed no signs of weakness either during life or after death; (2) those which, though weak, had no pulmonary murmur during life, but showed evidence of dilatation or degeneration after death; and (3) those in which a pulmonary murmur existed during life. (In some of these a mitral or tricuspid functional murmur was also present, but in no case was there any diastolic murmur).

The method employed was as follows. It was first noted whether any abnormal sounds arose when the pericardium was intact; then a small portion was cut away over the pulmonary artery and the heart again auscultated. Still more was then removed till the dorsum of the artery and the conus arteriosus bulged into the gap, when the observation was repeated. Lastly, the whole pericardium was removed and a final auscultation made.

Generally, the results obtained were these: 1. Healthy hearts with the pericardium intact gave no murmur; when it was removed from the pulmonary artery and conus at first there was no murmur and often none arose even though the heart was subjected to repeated compression for a long time, but, as a rule, sooner or later, especially if the heart were kept a day or so, bulging of the unprotected portions arose and was accompanied by a murmur. On removing the pericardium entirely, though the heart as a whole greatly dilated, sometimes to twice the previous size, yet no murmur was detected. 2. Hearts in which no murmur was noted during life but which had undergone dilatation and, perhaps, some fatty change. Still no murmur occurred with the pericardium intact unless the pressure used were very great or the heart had been dead a long time (from two to four days), but on partial removal of the peri cardium one nearly always occurred, and if it had been previously present it was increased, this increase again lessening or vanishing when entire removal of the pericardium was effected.

3. In the third class of hearts, where a murmur existed during life, this was always found with the pericardium intact, it

increased with its partial removal, and reverted to its previous character on total removal of the pericardium. The murmur thus heard bore usually the characteristics of that heard during life. It was harsh and blowing, was heard loudliest sometimes over the pulmonary trunk, sometimes over the conus; it was never well conducted, and, though generally audible over the aorta, its loudness was greatly diminished there. Even with healthy hearts in an intact pericardium this murmur could always be produced by pressure, amounting to indentation, on the dorsum of the pulmonary; but whereas in healthy hearts this indentation had to be considerable, when the artery was already dilated far smaller indentation would induce a murmur. It was always very difficult to produce such a pressure murmur in the aorta, and on no occasion did a murmur arise in this vessel if the left heart alone were distended with fluid and compressed in the usual way. On the contrary, if the right side only were distended the pulmonary murmur arose as readily, or even more readily, than when both sides were distended.

Experiment 3.—The patient, a male, died from cystitis and suppurative nephritis. The condition of the heart before death was not known. After death all the four cavities were dilated and the left ventricle was hypertrophied. There was some fatty degeneration of the muscle and some atheroma of the mitral and aortic valves and of the aorta and both coronaries. Pericardium intact: a short systolic murmur was best heard over the conus, but was also audible over the whole of the right ventricle, over the pulmonary valves, and the pulmonary artery; it was heard at the commencement of systole only, disappearing as soon as the artery was fully filled, and did not reappear so long as a steady stream was maintained. Pericardium removed from the artery: the pulmonary artery at once bulged forward and to the left and became much dilated. In addition to the above systolic murmur the *second* part of the systole was also accompanied by another murmur, this latter becoming continuous when a steady stream was maintained. The pericardium was now cut away from the conus as well and

this at once bulged prominently into the opening. Precisely similar but louder murmurs were again heard. Pericardium entirely removed: the sounds were the same as with the pericardium intact, but the continuous murmur could be at once reproduced by (considerable) indentation of the pulmonary artery.

Experiment 4.—A woman, aged 34 years, died from gangrene of the leg; no account of the heart was obtainable before death. After death the kidneys were found to be granular; the whole heart was much enlarged, the walls of both ventricles showing extensive fatty degeneration—that of the left ventricle was three-quarters of an inch thick, whilst the muscle layer of the right ventricle only measured $\frac{1}{32}$ of an inch. The valves of the right side were healthy; those of the left were thickened. Percardium intact: no murmurs. Pericardium removed from the pulmonary artery and conus: short systolic murmur. With pericardium intact there was also a short systolic murmur if stethoscope indented the artery or conus.

Experiment 5.—Heart from a man, aged 39 years. Death occurred from pneumonia of the right upper and middle lobes. The temperature averaged 103°F. The specific gravity of the urine was 1020 with a cloud of albumin. The heart's apex was in the normal situation, but the impulse was feeble; there were no murmurs. After death the heart was soft and flabby; the muscle showed cloudy swelling. Pericardium intact: no murmur. Pericardium partially removed; murmur appeared *at once.* This case shows the tendency pneumonia has to especially weaken the conus and pulmonary artery.

Experiment 6.—Heart from a youth aged 19 years who had suffered from leukæmia and purpuric hæmorrhage with constant pyrexia, averaging 101° F. Red corpuscles, 1,890,000; white corpuscles, 206,000; hæmoglobin, 30 per cent.; specific gravity, 1035. The heart's apex was normal in situation, but pulsation was most marked in the fourth space. There was vertical dulness at the upper border of the third cartilage, and right dulness at the left edge of the sternum. There were soft systolic

murmurs at the apex and in the pulmonary area. The spleen and liver were enlarged. Pericardium intact: distinct systolic murmur over the pulmonary artery and conus, heard best just over the valves; it shortened on raising the pressure of the fluid in the left heart. This is the heart I have shown you distended with paraffin, therefore I can give you no description of its condition. On removing the pericardium from the artery only, the murmur is increased but, if anything, shortened. The artery has a distinct crescentic antero-sinistral bulge over which the coat seems thinned. The murmur is very well heard over the upper two-thirds of the right ventricle. On further removing the pericardium from the conus a distinct bulge of the conus is noted, even before any distension of it is produced. Now the murmur is louder than when the pericardium was intact.

Experiment 7.—A woman, aged 35 years, who had suffered from obstructed common bile-duct. Much hectic fever before operation; soon after operation she had an obstructed umbilical hernia for which no operation was permitted and which caused death. This heart was experimented upon four days after death in hot weather. It was somewhat decomposed and both valves were dilated. The muscle was pale and there was much fatty infiltration. Only the left heart was used to see if the aorta uncovered by pericardium could be dilated by pressure in a manner similar to the pulmonary artery. It was subjected to a pulsating pressure of 140 millimetres, but no dilatation of the aorta could be detected.

Experiment 8.—A woman, aged 50 years, who had suffered from chronic focal myelitis. After death the kidneys were granular, the coronaries were thickened, and the heart was fatty. No murmurs were mentioned as having been heard before death. At first the double circulation was well maintained with a pressure of 150 millimetres on the left and 80 millimetres on the right, but 24 hours later, whenever the circulation on the left side rose much above that on the right, the circulation in the latter ceased. This was no doubt owing to bulging of the septum towards the right and so occluding the cavity of the conus.

Naturally this could not occur during life, as then the septum is not inert but actively contractile. Pericardium intact; no murmur. With pericardium removed from the pulmonary artery only, a systolic murmur arises. This remains unchanged when the pericardium is also removed from the conus, but vanishes with the complete removal of the pericardium. If free exit was given to the fluid from either aorta or pulmonary artery, so that these collapsed during diastole, then in *either* vessel a murmur arose with their re-distention in systole. This indicates the power a flabby vessel which cannot contract firmly on its contents possesses of producing a murmur. Is this the cause of the murmur which occasionally arises in the pulmonary area immediately after a large hæmorrhage?

Experiment 9.—A man, aged 72 years, who suffered from fracture-dislocation of the cervical spine. No observations were made before death. After death the heart was much decomposed and somewhat dilated. Pericardium intact: systolic murmur over the pulmonary artery and conus. Pericardium removed from the pulmonary artery and conus: the systolic murmur is longer and louder, but the change is not marked.

Experiment 10.—A man, aged 29 years, who died from carcinoma of the liver. There was nothing abnormal in the heart before or after death. Pericardium intact, no murmur.

Experiment 11.—The patient was a man who died from depressed fracture and cerebral hæmorrhage. No notes of the heart were taken before death; after death the heart was healthy but much decomposed. Pericardium intact; no definite murmur. Pericardium partially removed; definite systolic murmur.

Experiment 12.—The patient was a man who died from general tuberculosis. Before death no note was taken of the heart; after death all the cavities were dilated and the muscle was pale and flabby. Pericardium intact; no definite murmur. Pericardium removed from pulmonary artery and conus; quite definite systolic murmur. Pericardium also removed from aorta; systolic murmur less well heard. Pericardium entirely removed; sounds same as with it intact.

Experiment 13.—A woman, aged 40 years, who died from fractured jaw, gangrene, and pneumonia. No notes were taken concerning the heart before death. After death it was very flabby, but fairly muscular and of moderate size. Pericardium intact; a feeble systolic murmur was heard, but when the peripheral resistance was too small to keep the pulmonary artery distended then the murmur became loud and harsh, especially during the first half of systole before the artery was fully distended. The murmur could be heard over the aorta and conus but was only feebly conducted. Pericardium removed from pulmonary artery; murmur became more marked and longer. Pericardium removed entirely; murmur almost vanished. There may have been a pulmonary systolic murmur in this case before death as there was pneumonia, which puts special strain on the right ventricle; but, if not, the heart was very weak and no doubt acted feebly before death at a much less pressure than that to which we subjected it; our greater pressure was sufficient to produce dilatation with its consequent murmur.

Experiment 14.—The patient was a man, aged 50 years, who suffered from subacute nephritis. For two days after admission there was a distinct pulmonary murmur; this afterwards vanished* After death the liver was somewhat fatty and there was much general œdema. The coronaries were much thickened, the tricuspid valve measured five and a quarter inches in circumference and the pulmonary four and a half inches; the wall of the left ventricle was seven-eighths of an inch in thickness, that of the septum was three quarters of an inch, and that of the right ventricle from one-quarter of an inch to three-sixteenths of an inch. The pulmonary artery appeared considerably dilated, but the conus not more than the rest of the right ventricle and not so much as the right auricle. Pericardium intact; systolic

* This is interesting as showing the effect of rest from short exertions on the cause of the pulmonary murmur, for the man was too ill on admission to have lately undergone any lengthened labour. Hence he had nō mitral leakage; and, with the relief from any special strain given by rest, he soon ceased to have a pulmonary murmur, though the artery remained weakened and easily dilatable.

murmur was heard but no diastolic. Pericardium removed from pulmonary artery or from whole heart produced very little difference in the nature of the systolic murmur, but the experiment was continued for a short time only so that not much stretching could occur.

Experiment 15.—The patient was a youth, aged 16 years, who suffered from acute anæmia following a blow on the head eight weeks previously. On August 13th the red corpuscles equalled 770,000, and the white corpuscles equalled 2000; hæmoglobin equalled 15 per cent. On August 27th the red corpuscles equalled 450,000, and the white corpuscles equalled 2000; hæmoglobin equalled less than 10 per cent. Red cells varied in size and outline, but there was no marked poikilocytosis; the white cells were chiefly lymphocytes. The apex beat was slightly internal to the left vertical nipple line in the fifth space; there were systolic murmurs in the mitral and pulmonary areas. There was a venous hum in the neck. After death there were marked emphysema, œdema, and collapse of the lungs. There were some submucous hæmorrhages in the alimentary tract and the solitary glands were much enlarged. The heart's apex was in the left vertical nipple line and the fourth space; the pulmonary valves lay beneath the middle of the first left space. The pulmonary artery was not markedly enlarged, but the tissue of the conus was fatty. Pericardium intact; a systolic murmur was heard best on the left side of the pulmonary artery, next best on the right side, and scarcely at all on the dorsum. As the compressions continued (and dilatation increased) the murmur grew louder. Pericardium removed from conus and artery; the murmur was increased and continued to increase as the heart continued to give; it was still heard best in the same situations and scarcely at all over the aorta. Pericardium quite removed; heart dilated greatly, but the murmur grew less loud.

Experiment 16.—The patient was a man, aged 47 years, who suffered from hepatic cirrhosis, œsophageal hæmorrhage and ascites. After a severe hæmorrhage an apical systolic murmur appeared, which lasted a week, then vanished, and did not

reappear.* The apex beat was in the fourth space one and a quarter inches without the left vertical nipple line. There was vertical dulness up to the lower border of the second space. There was right dulness at the left edge. A systolic murmur was heard in the first four left spaces and slightly at the apex beat, but chiefly in the second and third. After death the apex beat was in the fourth space three and half inches from the middle line; the right edge was one and a quarter inches from the mid-line (opposite the fourth cartilage). The heart was enlarged and fatty; there was some atheroma, especially of the coronaries. Pericardium intact; a slight systolic murmur. Pericardium removed from pulmonary artery and conus; systolic murmur more marked. Pericardium entirely removed; systolic murmur much less distinct.

Experiment 17.—The patient was a man, aged 31 years, who suffered from acute pneumonia. Heart dulness was normal and there were no murmurs. After death the heart was seen to be very large; the tricuspid measured six inches and the pulmonary artery four inches. The wall of the left ventricle was fatty and there was some myocarditis. Pericardium intact; no murmur. Pericardium removed from conus and pulmonary artery; distinct but soft murmur. Pericardium entirely removed; a much less distinct murmur.

Experiment 18.—The patient was a man, aged 32 years, who suffered from fractured base. This was the same heart as that used in Experiment 2. It was macroscopically healthy, but as the experiment was performed eighty hours after death considerable decomposition and weakening of the walls had no doubt taken place. Pericardium intact; at first a soft murmur was heard both over the pulmonary artery and the aorta; it was then noted that there was a bulge of the pulmonary artery on its aortic side where the pericardium had been torn away; the pericardium was drawn over this bulge and stitched together, then this murmur vanished. Pericardium removed from conus and

* The patient was confined to bed, *vide* Experiment 14. If he had been up and about no doubt the murmur would have continued.

pulmonary artery; the usual loud systolic murmur was heard over the pulmonary artery and the aorta. Pericardium removed entirely; no murmur heard.

Experiment 19.—The patient was a man, aged 25 years, who died from a fall of forty feet. There was no disease of any organ. The heart was large but in good condition. Pericardium intact; no murmurs. Pericardium removed from conus and pulmonary artery; no murmur, but first sound became impure.

Experiment 20.—The patient was a man, aged 23 years, a draughtsman, who suffered from subacute Graves's disease and who died from shock two hours after removal of the goitre. Before death the apex beat was in the fifth space (half an inch external to the left vertical nipple line). There was vertical dulness in the upper border of the fourth cartilage, and right dulness at the left edge of the sternum. There was a systolic murmur at the lower border of the third left space, loud and harsh, rough and short; it was well conducted upwards on both sides of the sternum and reached the vessels of the neck and was much increased by pressure of the stethoscope. Only a partial *post-mortem* examination was allowed. The organs were removed through the wound. The lungs, the pleuræ, and liver appeared to be healthy; no adhesions were noted in the chest or the abdomen. The heart also seemed healthy, the wall of the left ventricle being in remarkably good condition, but that of the right ventricle was thin. With pressures of 80 and 200 milimetres the following results were obtained. With the pericardium intact a harsh and loud systolic murmur was heard, chiefly over the conus; it was heard very slightly in the pulmonary artery, no better, in fact, than it was in the aorta. Also a loud murmur was heard in the same situation throughout the diastole; this was when the venous (diastolic, pressure in supply pipes) was 50 on each side and it became faint on lowering this pressure to 15. If the pressure in the pulmonary circuit was increased, the systolic murmur was intensified, and when this pressure reached 200 its loudness

was doubled. It was noted that as the pulmonary pressure approached that in the aorta the systolic murmur was better conducted to this vessel. On removing the pericardium from the aorta the audibility of the systolic murmur over this vessel was lessened. On removing it from the pulmonary artery and conus the systolic murmur became louder and was well heard all along the pulmonary artery, but it was less well heard in the aorta, especially if the difference of pressures was maintained. With the pericardium entirely off the systolic murmur was as when the pericardium was intact, except that it was better heard along the pulmonary artery.

Experiment 21.—The patient, a man, aged 31 years, died from acute anæmia of four weeks' duration. The liver and spleen were enlarged. The apex beat was at the fifth rib and the left vertical nipple line. There was vertical dulness at the upper border of the third cartilage and right dulness a little to the right of the left edge of the sternum. Systolic murmurs were heard over the apex and pulmonary area and a soft systolic murmur, probably conducted from the left, in the second right space. After death the heart was flabby but not much dilated. The tricuspid valve measured $5\frac{5}{16}$ inches, the aortic valve $3\frac{1}{4}$ inches, and the pulmonary valve $3\frac{1}{8}$ inches in circumference. Scattered throughout the aorta and reaching up into the innominate and left carotid were remarkable bosses, almost ulcerated in places, of acute inflammation (hæmorrhagic aortitis). Pericardium intact: systolic murmur was heard; this was usually well heard in the aorta, sometimes so well heard there that it was doubtful in which vessel it arose [cf. condition of aorta and murmur of aortic degeneration]. Pericardium removed from conus and pulmonary artery: the murmur was much increased, but more over the pulmonary artery and conus than over the aorta. Pericardium entirely removed: murmur very feebly heard. Pressure on the pulmonary artery much more easily induced a murmur than similar pressure on the aorta even when the tension in both vessels was the same.

I trust that I have convinced you of two things: first, that in

cases of cardiac debility there is often a localised dilatation of the conus arteriosus and pulmonary artery; and, second, when in these cases a murmur arises in the second and third left spaces this murmur is due to such localised dilatation. But before I finish, though I have done what I set myself to do, yet I would like to point out several *à priori* reasons why we should expect such dilatation to take place. Debility is not a local but a general disease; the whole body is enfeebled owing to an insufficiently energetic metabolism. The heart also suffers as a whole, no one portion of it being more enfeebled than the rest. The weakness of the left ventricle is shown by the small and feeble pulse, incontrovertible evidence that the ventricular cavity is only partly emptied at each systole. But this does not mean any special failure on the part of the left ventricle; there is nothing to show that it is unable to keep pace with the lessened needs of the debilitated body. The body and ventricle fail together and equally, and hence there is no reason why the ventricle should show signs of strain. Both ventricle and body will tire soon; they are incapable of continuous toil, mental or physical, but they get tired together and so neither suffers undue strain.

If this is the case with the left ventricle, why should it not be so also with the right? It would be if everybody were debilitated; if we lived in a land where it was always afternoon, a land of sloth and idleness. But the surroundings of the sufferer from debility are too much for him; he cannot avoid catching somewhat of the spirit of emulation; moreover, this same debility renders his nervous system more irritable, less capable of self control, so he tries to keep pace with the healthy ones around him and, however much he may strive to take life easily, he must work against time now and then. The schoolboy is told to do as many sums as he can in an hour, he has to rapidly answer questions passed from one to another, he plays games which ever and again necessitate sharp bursts of exertion. The housemaid has to run upstairs quickly to answer her mistress's bell, is bound to carry heavy trays and scuttlefuls of

coal—all these short strenuous exertions are a healthful stimulus to the normal constitution, but to the debilitated they come as a breathless and exhausting labour. It is true he cannot keep them going for any length of time; he soon is obliged to leave them undone or finish them very slowly; thus he saves his left ventricle and body generally. In course of time experience teaches him perhaps to forego attempting their initiation, but experience, though an excellent, is yet a costly teacher; often, especially if the patient be young, the mischief is done ere the lesson is learnt. On what part of the body falls this mischief? What portion has to sustain the brunt of the shock of any sudden exertion—has to play the buffer to the rest? I think it is the lungs and next the right ventricle.

Physiologists* tell us that the first result of exercise is an increase in the rate and depth of the respirations—that is, of the respiratory exchange. That the respiratory quotient, $\frac{CO_2}{O_2}$ is not increased, but if anything diminished; in other words, the tissues are as rich (or richer) in oxygen during exercise as during rest. This necessitates a great increase in the absorption of oxygen at this time, for E. Smith has shown that a man gives off ten times as much carbolic acid when on the treadmill as he does when asleep. But what I would particularly draw your attention to is this—that arm work, *per unit of work done*, requires a greater absorption of oxygen than climbing, and climbing than walking on the level. If the amount of oxygen absorbed during sleep per minute be 100 grammes, then in a minute's walking at three miles an hour on the level it would be 500 grammes; in climbing a yard high 5,000 grammes, and in doing the same amount of kilogrammetres by turning a wheel (arm work) 7,000 grammes.

Such an enormous increase in the absorption of oxygen and giving out of carbonic acid must seriously strain the resources of the organs concerned. The lungs are rarely at fault, for they have so much unused tissue which they can bring into play

* Pembrey in Schaefer's Physiology, vol. i., pp. 714 et seq.

and so many extraordinary muscles that they rise equal to almost any emergency.

This increase in pulmonary action is probably brought about by some product of muscular activity stimulating the respiratory centre of the medulla, but such stimulation would be of small avail were not the right ventricle similarly stimulated to supply the lungs more rapidly with blood. Such a stimulus is provided by the extra flow of blood from the veins, this being induced by the aspirating action of the increased inspirations. Thus, as regards the circulatory system, on the right ventricle and pulmonary artery falls the first brunt of the extra labour; and when this is undertaken suddenly, as in a run upstairs, the shock it gives to these organs must be extremely great.

It is true that the extra toil must soon fall on the left ventricle also, but it does so more gently and with far less suddenness, for (*a*) the extra blood can only reach it through the devious elasticity and dwindling pressure of the pulmonary system; (*b*) the systemic circulation in debility is but partly full, so that several systoles are occupied in properly filling this, and thus the left ventricle is allowed a short period in which to brace itself up for its extra work; (*c*) if it be unequal to this extra work it can easily refuse to receive all the blood sent to it, for it is no strain on a high-pressure cylinder to reject fluid sent it from a low-pressure one through elastic tubing; (*d*) if it does so reject the blood the great dyspnœa thus excited soon puts an end to the patient's exercise and thus at once eases the left ventricle, whilst the blood thus blocked in its flow through the lungs must be a terrible additional shock to the right heart and pulmonary artery; (*e*) in any case general exhaustion soon stops any sharp, strenuous toil on the part of a sufferer from debility, hence the left ventricle is not likely to be severely taxed.

Again, the increase in respiration must increase the difference in the intra-thoracic pressures in inspiration and expiration; this may be from five to ten millimetres Hg. Such a difference,

rapidly produced and constantly repeated, must have a considerable dilating influence upon an artery so distensible as the pulmonary, whose capacity, according to Roy, is increased twelve times by raising the pressure from 0 to 36 millimetres Hg.

As a negative proof of this contention let me draw your attention to a case where a debilitated subject was carefully guarded from all these sudden exertions, and where the heart failed, it is true, but failed in a very different manner from that which I have been describing.

Case 31.—The patient was a married woman, aged 36 years. She was never very strong; during the last week she had not been as well as usual. She was admitted into hospital immediately after a large hæmatemesis—more than one pint. There were no murmurs on admission; the pulse was hæmorrhagic with occasional dropped beats. Examination of the blood on admission revealed red corpuscles, 1,560,000; hæmoglobin, 25 per cent.; and white cells, 13,000. One month later, with the patient still lying in bed in the hospital, there was no pulmonary murmur, but both a mitral murmur and a tricuspid murmur. The apex beat reached the fourth space just within the left vertical nipple line. There was vertical dulness at the middle of the third space and right dulness half an inch to the right of the right edge of the sternum. This was a very different outline from that which I have described to you as occurring in debility. The blood, though better, was still poor—viz., red corpuscles, 2,800,000; white corpuscles, 10,000; hæmoglobin, 30 per cent. Here the heart had shared in the debility arising from a severe hæmorrhage in a weakly subject, but no part of it had been subjected to more strain than the rest. The patient had been kept in bed on a low diet apart from all domestic worries. Her physical exertion was of the smallest possible amount and what she did could be done as slowly as she pleased. Hence both ventricles underwent a general dilatation, allowing leakage through both auriculo-ventricular valves. If she had been up and about endeavouring to go on with her maternal and

housewifely duties I have no doubt in my own mind that her cardiac outline would have been typical to a marked degree of that characteristic of debility, whilst a pulmonary murmur would not only have been present, but would have been the chief of the three; but, as it was, the heart gave no evidence at all of any special enlargement of the conus or of a pulmonary murmur.

Such cases as this, as well as those previously mentioned which showed no pulmonary murmur, though not seldom a mitral as long as the ill-nourished frame was kept at rest in bed, but in which one often arose when the patient was allowed to be up,—such cases seem to me to be exceedingly strong evidence in favour of the view that it is sudden sharp exertion which produces the peculiar localised failure of conus and pulmonary artery.

It is, of course, quite common for hearts of debilitated subjects who are leading an active life to give way altogether and allow tricuspid and mitral leakage, but when they do they never fail to show the large increase in the vertical dulness which is absent in the case just described; the general giving way is an addition to the special and initial failure; the vertical dulness remains raised, but the right and left dulnesses become increased also.

If, as I have tried to show, the right ventricle should be the first to yield, it is not difficult to understand what part of it should first give way.

We may consider the whole ventricle as one half of a cone which has been bisected from apex to base (Fig. 12); the stout septum ventriculorum forms the triangular surface of section, and as the left ventricle does not fail, no more will this septum; hence we may look upon the ventricle as a triangular board to which is attached a semicircular convexity of rubber; it is evident that this yielding rubber will stretch most where it is least restrained by the rigid triangle*—viz., along the semicircular border of its base. Also that the portion of this border which

* This helps also to show why the enlargement of the right ventricle in mitral disease assumes a different form, for in this case the septum has already become itself stretched along with the rest of the left ventricle.

will stretch most will be the conus, for the lowest part is held in check by the unyielding, rigidly fixed inferior vena cava and the middle portion by the chordæ tendinæ of the tricuspid valve

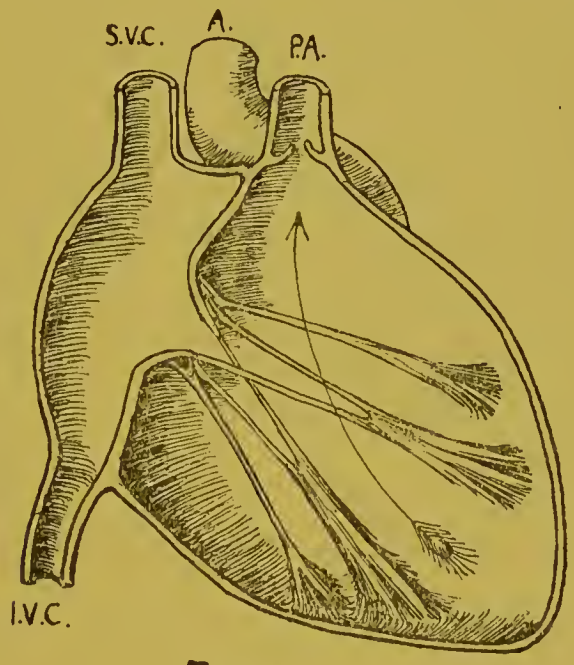


FIG. 12.

and their stout musculi. Further, the direction of the blood-current is from the apex towards the pulmonary valves; the conus will tend to give, therefore, most in the direction where it is longest distended with blood.

Given this dilatation of the conus that of the pulmonary artery is easily explained. The dilating conus carries the valves upwards and thus shortens the length between the two extremities of the artery. The artery becomes lax, and when distended with blood enlarges in its transverse diameter, so that its wall may occupy the same superficial area. Finally, we must remember the easy distensibility of the pulmonary artery, which is proportionately, after due allowance is made for the different tensions at which they work, some six times that of the aorta.

I have forborne all reference to authorities. I trust that this will not be put down to any lack of respect to other workers in the same field of inquiry. It is simply because I found them to be so many and so important that if I had once begun to quote

and, necessarily, often to combat others' views, there would have been no time left to place my own before you.

I have finished, and if these views do not commend themselves to your judgment believe that this want of conviction is due to my feebleness of exposition and not to their lack of truth. But at least I trust that this lecture will give some impetus to the study of this common and important cardiac condition, and that we shall not rest till not only this but all allied functional cardiac states are settled to our individual satisfaction and our text-books speak with no uncertain sound both of their phenomena and clinical importance. For myself I trust in subsequent papers to still further elucidate such matters as are germane to the discourse which it has been my high privilege to deliver this afternoon.

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